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EXPERIMENTAL PULMONARY ANTHRAX IN MICE THROUGH INTESTINAL ENTRY

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EXPERIMENTAL PULMONARY ANTHRAX IN MICE THROUGH
-INTESTINAL FATRY

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EXPERIMENTAL PULMONARY ANTHRAX IN MICE THROUGH

INTESTINAL ENTRY

/Following is the translation of a French-language scientific paper by H. Velu, P. Soulie and R. Courtade in Comptes Rendus de la Societé de Biologie, (Reports of the French Biological Society), Vol. 135, Paris, 1941, pp 1354-55.7

In spite of the almost insurmountable resistance to anthrax infection by the ingestion of food contaminated by spores as shown by the guinea pig and the rabbit, the alimentary origin of anthrax in domestic animals is admitted by all pathologists since the work of Pasteur, Chamberland and Roux on the one hand and R. Loch, Gaffky and Loeffler on the other. Opinion differs only as to the place and mode of entry of the causal agent. Pasteur et al assumed that a trauma of the digestive tract is required to trigger infection; Koch adopted the supposition of germination of the spore within the intestinal cavity; Sanarelli bases himself on inhelation and passage from the pharynx into the respiratory tract.

The hypothesis of Besredka on the elective receptivity of the cutaneous system induced Sanarelli (1), A. Boquet and A. Saenz (2), Hruska (3) to resume investigation of the digestive permeability in the guinea pig. Their research tends to prove that germination of spores does not exist within the intestinal tract of laboratory animals.

A Poquet has shown that the ingestion of anthrax spores by the guinea pig after fasting for 36 hours is followed by their transition into the blood circulation during digestion and that they may invade the entire circulation without inducing either infection or immunity. However, when the skin is injured while the blood carries the spores, the guinea pig (4) and infant goat (3) may succumb to anthrax. In that case, we observe at the point of injury which may be no larger than a pin-prick, an endogenous local anthrax which results from the diffusion of bacilluscontaining blood around the injured integument.

We have tried to provoke pulmonary anthrax in the same manner in five groups of five mice each. Groups a and b were given drinking water for 48 hours containing a suspension with 1.5 billion spores per ccm of which the mice absorbed a quantity of 2 ccm per day. After 48 hours, group b was subjected to irritation by chlorine of the respiratory tract at the same time as group c which was previously exposed to the effects of an aerosol containing spores of which about 200 were retained in the pulmonary parenchyma. The chlorine titer of the irritating atmosphere was very low, barely 1,500 as expressed by C x t (C = overall concentration in

milligram per cubic meter; t = exposure time in minutes). Group d was subjected exclusively to the inhalation of spores.

In group a subjected exclusively to ingestion, there was no case of mortality; in the group b (ingestion — chlorine), 3 out of 5 mice died of pulmonary anthrax, one after 48 hours and the other 2 after 56 hours. In group c (inhalation — chlorine), 4 mice succumbed to anthrax, 2 after 48 and 2 after 72 hours. All mice of group d (inhalation only) survived as well as all those of group e (inhalation of chlorine only). The dosage of chlorine employed is therefore clinically inactive in mice and this may explain the survival of 2 mice in group b and of 1 in group c. These results are similar to those obtained by S. Arloing et al (5) in their work on the action of chronic intoxication by inhalation of chlorine at low concentration for inducing experimental tuberculosis in the guinea pig.

Summary:

- a) In addition to chalk dust held responsible by S. Lodge (6) as the cause of pulmonary anthrax in workers of the wool industry, we should include the widely used chlorine (for bleaching fibers and processing salvaged wool) as a contributory agent even if the entry of the anthrax spore takes place through the intestine.
- b) When slightly irritated by the inhalation of clinically inactive chlorine, only the lung becomes receptive for the anthrax spores, regardless of the point of introduction, in the same manner as the skin slightly injured by a pin-prick and perhaps to a greater extent because the receptivity of the skin is not as pronounced as is generally believed. Incculation of anthrax by cutaneous scarification is often a failure if the matter to be tested contains only spores.

References

- 1 Sanarelli. Ann. de l'Inst. Pasteur, 1925, t. 39, p. 209
- 2 A. Boquet and A. Saehz. Ann. de l'Inst.Pasteur, 1933, t. 50, p. 320.
- 3 C. Hruska. C. R. del'acad. des sc., 1928, t. 186, p. 1868.
- 4 A. Boquet. C.r. del'Acad. des sc., 1924, t. 178, p. 260.
- 5 F. Arloing and collab. Presse medicale, no. 33-34, 10 April 1940, p. 361.
- 6 S. Lodge. Arch. de med. exper., 1890, p. 759.

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